



Chronic intramuscular calcific tendinitis of the deltoid muscle

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Abstract

Calcific tendinitis is a potentially symptomatic disorder characterized by calcium deposits in the substance of the tendon. Although this condition can occur in any tendinous tissue throughout the human body, calcium deposition commonly occurs at tendon insertions near the bone–tendon junction. The musculotendinous junction of the deltoid muscle has peculiarly dense intramuscular tendons to which muscle fibers attach obliquely to create muscular strength. Given that the intramuscular tendons themselves, which form the consecutive part from the insertion, are subjected to unpredictable stress load or microtrauma similar to tendon insertions, it is reasonable to assume that calcific tendinitis could also occur at the intramuscular tendons. Here we report a case of chronic symptomatic calcium deposition in the lateral part of the deltoid muscle between the origin and the insertion, which was eventually surgically removed and confirmed as intramuscular calcific tendinitis.

Keywords Calcific tendinitis · Intramuscular tendon · Deltoid muscle · Shoulder

Introduction

Calcific tendinitis represents the pathological deposition of hydroxyapatite crystals in tendons. On imaging examinations, the acute calcium deposits are observed as a fluffy texture with an ill-defined margin. These deposits mainly appear when the calcium is resorbed and are often painful. In contrast, subacute or chronic calcium deposits have homogeneous dense calcium deposits with well-defined margins. These phases indicate a formative or resting phase of the calcific stage and usually remain mildly symptomatic or asymptomatic [1, 2]. However, it is not always symptomatic and can be an incidental finding in any joint or periarticular soft tissues.

While calcium deposition can occur in any tendinous tissue throughout the human body, it commonly forms at the tendon insertion near the bone–tendon junction [3]. In the shoulder, this condition is typically observed the rotator cuff tendons but is uncommon in other sites [2, 4, 5]. The acute calcium deposits in the resorptive phase are

occasionally associated with the migration of calcium in the surrounding tissues and often extrude from the tendon into the subacromial space and bursa [6]. However, intramuscular migration of calcium deposits is uncommon and has rarely been reported [7–10].

Here, we report a case of chronic symptomatic calcium deposition in the lateral part of the deltoid muscle belly between the origin and insertion, which was eventually surgically removed and confirmed as intramuscular calcific tendinitis.

Case report

A 73-year-old female pediatric nurse experienced aggravated severe pain in her left (non-dominant) shoulder, which occasionally increased in intensity. Her first hospital visitation occurred at 67 years of age due to mild chronic persistent pain that had persisted for 3 months. At that time, she had no special family history, hereditary diseases, metabolic dysfunction, systemic disorders, abnormal blood examinations, a history of trauma, or local injections to the muscle. A physical examination revealed a mass in the lateral part of the left deltoid muscle. Although the mass was tender, no functional impairment was observed. X-ray revealed irregular fusiform calcium deposition in the lateral portion of the left deltoid (Fig. 1). Magnetic

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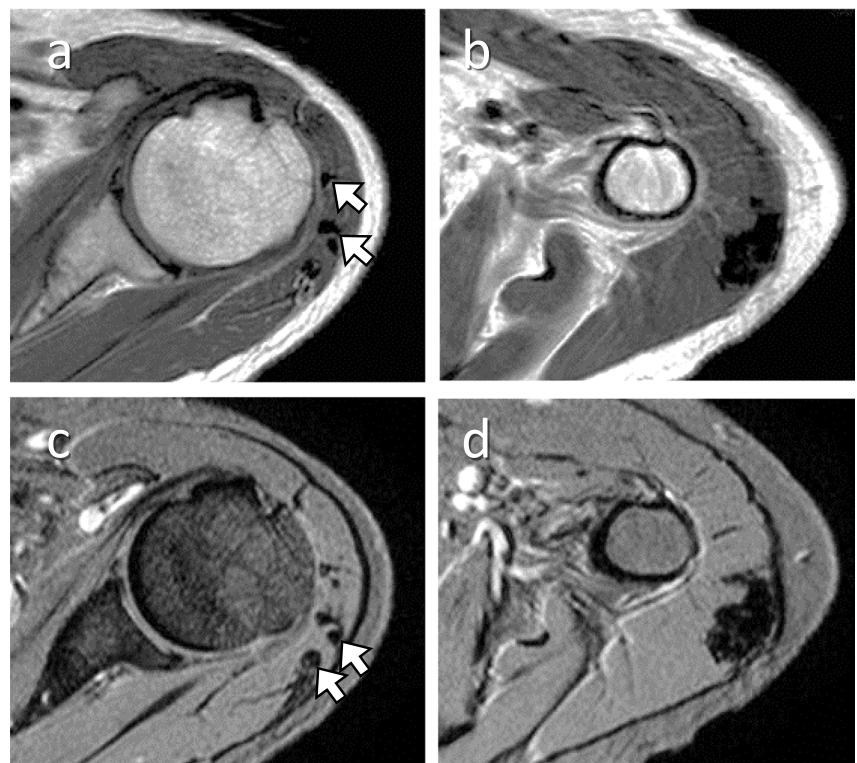


Fig. 1 X-ray at 67 years of age showing a calcified soft tissue mass with clear boundaries within the left deltoid region

resonance imaging at 1.5 Tesla was performed to evaluate whether the shoulder pain also came from the rotator cuff, even though there was no rotator cuff tear. The calcium deposits showed low signal intensity in the intramuscular tendons of the deltoid muscle in both T1-weighted and fat-suppressed T2-weighted fast field echo images (Fig. 2a–d). Pain was temporarily controlled using nonsteroidal anti-inflammatory drugs (NSAIDs), as required.

For 6 years, her symptoms were controlled with the conservative treatment, but the pain aggravated and X-ray revealed that the calcium deposits increased (Fig. 3). Computed tomography images were acquired using a 320-row computed tomography scanner with 5-mm slice thickness. The calcium deposits were approximately $35 \times 10 \times 10$ mm fusiform mass (Fig. 4a). Some of them were sparsely distributed in the distal and proximal parts of the deltoid muscle as a fluffy texture with an ill-defined margin and suspected of intramuscular migration (Fig. 4b, c). The pain did not improve after 2 months of conservative treatment with NSAIDs. Therefore, she was scheduled for surgical excision of the calcium deposits. Upon opening the muscular fascia during the surgery, milky calcium spouted out, after which a toothpaste-like, chalky-white matter and gravelly deposits were curetted and calcified intramuscular tendons were removed, which was subsequently confirmed as intramuscular calcific tendinitis by histopathological examination (Figs. 5a, b,

Fig. 2 a–d Axial T1-weighted (repetition time/echo time 652/11, fractional anisotropy 90°) (a, b) and fat-suppressed T2-weighted fast field echo (repetition time/echo time 880/14, fractional anisotropy 25°) (c, d) images showing low signal intensity in the calcium deposits located in the deltoid muscle. a, c Proximal slice of the island form of the intramuscular tendon (arrowheads) showing remarkable calcification. b, d Irregular calcified mass at the middle slice



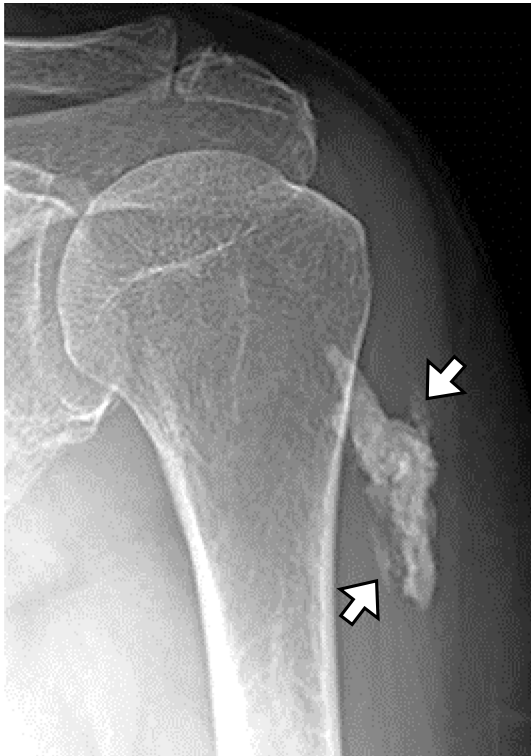


Fig. 3 X-ray at 73 years of age shows increasing calcium deposits. Some of the calcium deposits present fluffy texture with an ill-defined margin (arrowhead)

and 6). Following the surgery, her symptoms disappeared without functional impairment of the shoulder and no recurrence 2 years after the surgery.

Discussion

Calcium deposition is particularly frequent around the shoulder and mostly appears in the rotator cuff tendons [2, 4, 5, 11–13], although it can also be found at the acromial attachment of the deltoid [14] or the long head of biceps brachii tendon [15]. Although it can occur in other muscles (e.g., the gluteus medius, gluteus maxims, and longus colli), all calcium depositions form at the tendon insertion near the bone–tendon junction even though it can rarely form at the tendinous portion near the musculotendinous junction [16–18]. To the best of our knowledge, this is the first report describing the clinical manifestations, relevant imaging findings, and macroscopic observations associated with chronic intramuscular calcific tendinitis of the deltoid muscle.

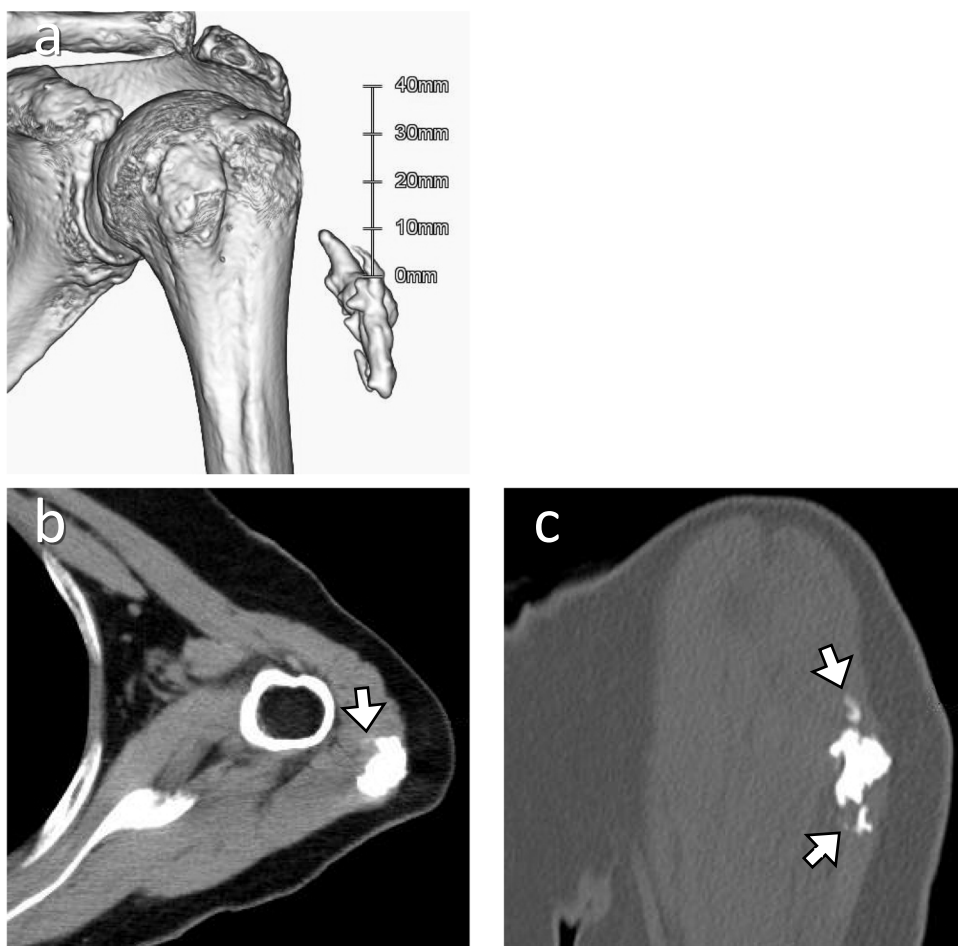
The pathogenesis of calcific tendinitis is still under discussion [8, 13, 19, 20] and several different theories have been proposed, for example, degenerative calcification following local ischemia or necrosis of tenocytes and reactive

calcification following fibrocartilaginous metaplasia [3]. However, the formation of intramuscular calcium deposits is hypothesized to be different because of the formation of hematomas after trauma or microtrauma [9, 10, 20]. This patient developed calcium deposits, which occurred in the intramuscular tendon of the deltoid muscle. The deltoid muscle has peculiarly dense intramuscular tendons to which many muscle fibers attach obliquely to create strong muscle contractions [21, 22]. The intramuscular tendons, which sustain the transmission of forces themselves, are subjected to unpredictable load stress or microtrauma, such as muscle strains [23, 24]. Tendon overuse might be a factor in the development of calcific tendinitis. It was reported that the supraspinatus tendon had increased expression of cartilage genes when the tendon was subjected to overuse in a rat model [25]. Hence, it is reasonable to assume that calcific tendinitis could also occur in intramuscular tendons of the deltoid even with no history of trauma in this case.

Sufficient blood supply to the surrounding muscle tissues allows for the resorption of the initial calcium deposits at the intramuscular tendon. The disappearance of the calcifications shortly after the resorptive phase is not uncommon and granulation tissue replaces the space left behind by the calcium deposits and eventually matures into a fibrotic scar [11]. However, when calcium deposits rapidly increase in size and do not show any signs of resorption, they can become chronic calcific tendinitis surrounded by a hard calcium shell. Although the patient had chronic dull pain, intramuscular migration of calcium deposits could have caused acute pain. Intramuscular migration usually occurs in the resorptive phase of calcific tendinitis and the migrated calcium deposits increase in size. As we observed on the X-rays when a symptom is aggravated, the migrated deposits are of a lower density than the stable portion of the calcium deposits that they migrated from, and their contours become ill-defined [4, 8, 26]. Therefore, more symptomatic flare-ups of chronic calcific tendinitis can occur occasionally. Additionally, the migrated deposits are known to be most dependent on the identification of local inflammation of the edema involving portions of the muscle that contain hypointense calcific foci on MR fluid-sensitive sequences [9]. Although additional preoperative imaging examinations were only performed with computed tomography to confirm the location of the calcium deposits, if MRI was performed again preoperatively, it might have reflected these findings observed in the intramuscular migration of calcium deposits.

Given the unusual nature of intramuscular calcific tendinitis of the deltoid muscle, it is important to consider the differentiation with other entities. X-ray is usually the initial imaging modality by which soft tissue

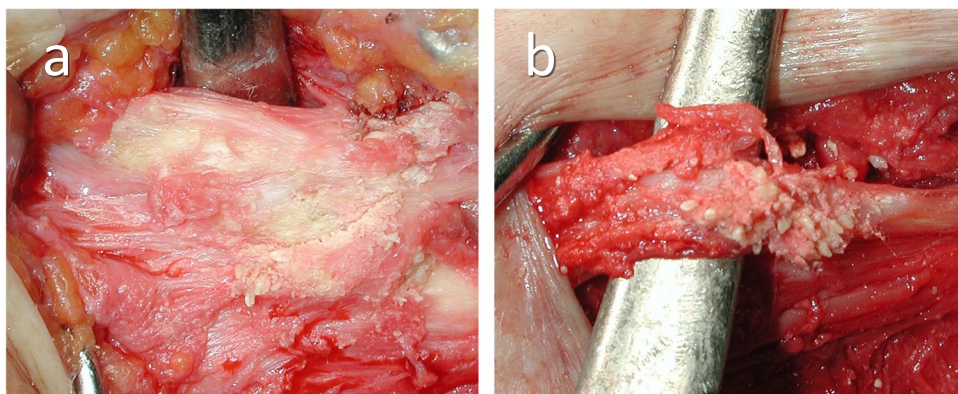
Fig. 4 a–c Three-dimensional (a), axial (b), and sagittal (c) computed tomography images revealing calcium deposits in the deltoid muscle. **a** Irregular fusiform calcific mass measuring 35×10×10 mm. **b** The ill-defined margin part wherein the resorption of the calcium deposits was observed (arrowhead). **c** Sparse distribution of calcification in the distal and proximal parts of the deltoid muscle



calcifications and ossification are detected. Calcifications normally appear as mineralized densities similar to this case, whereas mature bone shows an outer cortex and inner trabecular pattern [27]. In the computed tomography images, calcifications show slightly lower Hounsfield unit values than the bone [28]. Calcium deposits usually demonstrate low signal intensity on all MRI sequences. While other calcifications in the muscles,

such as tumoral calcinosis [27], myonecrosis [29], and calcified leiomyomas [30], often appear as heterogeneous lesions with mixed signal intensity, calcific tendinitis show uniformly low signal intensity. As there was no malignant finding on histopathological examination and it was a normal calcific tendinitis, we diagnosed it as intramuscular calcific tendinitis. Although it remains unclear whether calcium deposition occurs at the muscle

Fig. 5 a, b Intraoperative images (a, b) revealing calcium deposition in the deltoid muscle. **a** Gross appearance of the calcium deposits. **b** Presence of deposits along the intramuscular tendon



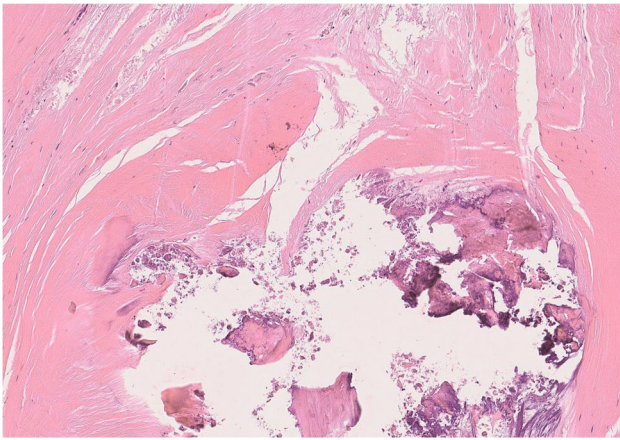


Fig. 6 Photomicrograph of the calcified tendon following fixation and decalcification. Histopathological examination showed intramuscular calcific tendinitis. Calcium deposits are observed in the lower right of the photomicrograph. Calcifications with fibrous degenerative changes in the collagenous fiber of the tendon indicate basic calcium phosphate (hydroxyapatite) crystal deposition. There is no evidence of malignant transformation

fibers or the intramuscular tendons, these conditions may be similar to intramuscular migration of calcium deposits [7–9].

As for the treatment of calcific tendinitis, conservative treatment is the first line of treatment and we chose NSAIDs to control the pain. Although mild chronic persistent pain was temporarily controlled using NSAIDs, the calcium deposits increased when her symptoms aggravated. Steroid injection is one of the options to control the acute pain of the calcium deposits, but the effects are controversial [19, 31] and it might result in negative effects in the form of stopping reabsorption of the deposits [32]. Other conservative treatments, such as ultrasound-guided needling and extracorporeal shock wave therapy, have been reported to produce good results when applied to both acute and chronic calcium deposits [5, 12]. Although these treatments might be effective, the calcium deposition observed in the deltoid muscle was a longstanding and extensive calcification. Moreover, intramuscular calcium deposits are thought to be unresponsive to conservative treatment, and good results have been reported with surgical excision [10]. Accordingly, this case was also treated by surgical excision and it has shown excellent clinical outcomes.

In conclusion, calcific tendinitis usually occurs at the tendon insertion near the bone–tendon junction. In rare circumstances, however, calcification can occur in the intramuscular tendons. The increase in the size of symptomatic calcium deposits without significant absorption may require surgical excision.

Declarations

Informed consent Informed consent was obtained from the subject described in this case report.

Ethical approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Conflict of interest The authors declare no competing interests.

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